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# Effect of Pleural Membrane on the Propagation of Rayleigh Waves in Inflated Porous Lungs—A Study

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**ABSTRACT** In an attempt to include the effects of natural porosity of lung parenchyma into the mathematical study of lung diagnostics, a model describing the propagation of low-frequency Rayleigh waves in relation to the porous architecture of the lung parenchyma is presented. The wave motion is analyzed by assuming that the lung parenchyma behaves as an isotropic elastic half-space containing a distribution of vacuous pores with the visceral pleura as a taut elastic membrane in smooth contact with the half-space. The thinness of the pleural membrane in comparison with the large surface area of contact enables it to be modeled as a material surface in contact with the parenchyma. Utilizing the perturbation technique, an approximate formula for the Rayleigh wave velocity in the parenchyma with allowance for surface tension, mass density, and porosity is derived. In addition, the effect of the tension in the pleural membrane and the porosity in the parenchyma on the propagation of the low-frequency Rayleigh waves is brought out through the dispersion spectrum. It is hoped that the results of this paper would enable a better understanding of the porosity and surface-tension effects on lung parenchyma.

**INDEX TERMS** Pleural membrane, porous lung, low-frequency, material with voids, Rayleigh waves.

## I. INTRODUCTION

The mechanical properties of biological tissues are known to provide information about their pathological condition and have been clinically used for diagnostic purposes in numerous organs [1]. Of all the internal organs, the lung has the strongest connection between physiologic function and mechanical behavior [2]. It is well realized that lung elastic recoil plays a crucial role in breathing and hence, lung mechanics, particularly elasticity during physiology, has received large attention in the literature [2], [3]. Techniques are being developed to contrast and quantify changes in the macroscopic properties of the lung that are indicative of disease and may be linked to behavioral and structural changes at the microscopic level [4].

A most common name for techniques developed to non-invasively assess the mechanical properties of biological soft

tissues with application to medical diagnosis is elastography [5]. A general approach in elastography is to perturb the state of the tissue under the study using a quasi-static, harmonic or transient mechanical source and then infer the biomechanical properties from the measured mechanical response using a model [6]. Current elastographic techniques enable elasticity estimation by determining the phase speed of shear/surface waves propagating in the tissue [7]. Shear/Surface wave dispersion derived from the elastography technique can be used to estimate the elastic/viscoelastic parameters with an assumed model of the tissue. In the model, the problem of wave propagation on the surface of the tissue is generally approximated as wave propagation in a semi-infinite elastic/viscoelastic medium under harmonic excitation.

The Rayleigh surface waves [8] have proven to be applicable in many areas such as ultrasonics, seismology, material science and medical diagnostics to name a few. A better understanding of the surface wave behavior on and in soft

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biological tissues is likely to aid medical diagnostic techniques [7]. A methodology for the excitation and propagation of surface waves on a viscoelastic half-space with application to medical diagnostics was presented by Royston *et al.* [9]. Zhang and Greenleaf [10] developed a noninvasive and non-contact method for the estimation of tissue's elasticity with surface speed. Research into the use of surface wave methods for the assessment of bio-mechanical properties of lung has resulted in models for estimating lung elasticity [11], estimating local viscoelasticity of lung [12], assessing superficial lung tissue diseases [13] and assessment of interstitial lung diseases [14].

The lung is an organ with complex internal structure that consists of a network of stiff airway tubes, bronchi and bronchioles embedded in a soft porous tissue called the lung parenchyma and enclosed by a thin pleural membrane known as the visceral pleura. The lung parenchyma whose primary function is gas exchange is a foam like structure composed of bronchioles, alveoli and the enclosed air volume. On a macroscopic scale that encompasses several alveoli, the parenchyma can be regarded to be a homogeneous structure that behaves isotropically [15], [16]. The mechanical properties of the lung parenchyma reflect its microstructure and play an important role in the functioning of the normal lung and the changes in these parameters index the pathology of the lung [17]. Evidence for the relationship between changes in parenchymal material properties and pathological processes can be found in a variety of lung diseases [18]. As the mechanics of lung is dominated by the parenchyma, modeling the parenchyma is crucial for lung diagnostics. Literature on the modeling of lung parenchyma is quite substantial and goes back to at least a century and a half. A brief review of the work relevant to this study is outlined here for completeness.

The mechanical behavior of one element or region of the lung is affected by its interaction with the rest of the lung. To understand this interdependence between the different lung units, Mead *et al.* [19], in a classical biomechanical study, illustrated the effect by introducing a lung model that comprises a two-dimensional hexagonal network of springs. Wilson [20] put this concept in a continuum mechanics perspective by showing that the hexagonal network of springs can be approximated by continuum mechanics models when parenchymal deformations are small and linear. Later Lambert and Wilson [21] pictured the lung parenchyma as a number of interconnected, random oriented, plane, elastic membranes called the conceptual model, and followed it by a mathematical model that comprises a set of equations that describe a continuum which is mechanically equivalent to the conceptual model. The elastic properties of the lung parenchyma associated with the model proposed by Lambert and Wilson [21] were experimentally reported by Hajji *et al.* [22]. The experimental results reported-on the values of the shear modulus of the parenchyma and the tension in the pleural membrane-were based on a mathematical solution for the indentation of an elastic half-space covered

by a membrane. It was observed that the shear modulus of the parenchyma is a function of the transpulmonary pressure and the fractional contribution of the pleura to the incremental work done by the lung under quasi-static conditions is estimated to be about 20% which was further validated by Stamenovic [23] and Suki and Hantos [24]. Lai-Fook [25] showed that a continuum mechanics based analysis of the deformation of the parenchyma surrounding the large pulmonary vessels can be used to interpret changes in vessel diameter resulting from variation in vascular and lung transpulmonary pressure. Based on this seminal work, most modeling studies of air way- parenchymal interdependence have considered the parenchyma to behave as a uniform and isotropic elastic continuum. Ma and Bates [26] have compared the continuum theory with spring network models of air way- parenchymal independence and have shown that the displacements and stresses produced in the parenchyma surrounding a contracting airway propagates farther from the air ways in the hexagonal network of springs model than in a continuum mechanics model.

The porous architecture of the lung tissue has motivated researchers to focus on multi scale modeling of the lung parenchyma. On a macro-scale, the lung can be modeled as a two-phase medium consisting of the solid and fluid phases. The solid phase includes the deformable thin walled tissue and the fluid phase consists of the air in alveoli and ducts and blood inside the walls [27]. Kowalczyk [27] proposed a mechanical model of lung parenchyma as a two-phase porous medium in which general constitutive relations for both phases and their mechanical interactions have been formulated. The formulation provided a numerical approach to non-linear quasi-static problem of deformation in the parenchyma. In the model, blood has been considered to be a component of elasticity of the solid phase. Lande and Mitzner [28] present a new approach that characterizes the dynamics of gas flow into a viscoelastic porous medium that models the lung structure. It was further shown how the loading impedance at the lung boundary may have a significant impact on the dynamic behavior of whole lung viscoelasticity. Current theoretical models for the acoustic properties of the parenchyma are usually based on the work of Rice [29], in which the parenchyma is modeled as a homogeneous mixture of a gas phase and a tissue phase [30] wherein the communication between the alveoli are considered to negligible. The model of Rice [29] is generally referred to as the bubble swarm model. Siklosi *et al.* [31] model the Parenchyma as a porous solid with air filled pores and consider Biot equations [32], [33] as a model for its acoustic properties. They have reviewed how homogenization can be used to derive the Biot equations from a model of the micro-structure of a porous material. It was also opined that Biot equations could provide a theoretical framework to explain the variations in the speed of sound observed experimentally in a parenchyma. In [34], the parenchymal structure was considered to be highly heterogeneous on a small scale  $\epsilon$  and two-scale homogenization techniques were used to derive effective acoustic equations

for asymptotically small  $\epsilon$ . The discontinuous Galerkin formulation was used to investigate the sound propagation in the homogenized parenchyma. Utilizing homogenization theory Cazeaux *et al.* [30] developed non-dissipative model of Rice [29] for the propagation of low-frequency sound in a domain modeling the parenchyma. They also investigate the asymptotic behavior of this medium as the size of the alveoli tends to zero. Gouldstone *et al.* [35] developed a multiscale multiphysics model of lung parenchyma that generates synthetic images of alveolar compression under spherical indentation at the visceral pleura of an inflated lung. A finite element mesh was used here for computational indentation on the macro scale. Dai *et al.* [4] compared the bubble swarm model with the model based on Biot theory of poroviscoelasticity [32] for the sound and vibration in the lungs by integrating a fractional derivative formulation of shear viscoelasticity into both the models. In the application of Biot theory, air was considered as the only fluid in the lungs. The study [4] suggests that the model based on Biot theory was more accurate than the bubble swarm model for wave propagation in the lung. Recently, an ultrasonic surface wave technique-ultrasound surface wave elastography – had been developed for assessing various skin and lung diseases [13], [14], [36], [37]. The technique provides a non-invasive and quantitative method to generate and measure the surface wave speed of skin/lung tissue and then estimate the tissues elasticity according to the measured wave speed using a theoretical model. The theoretical model consists in analyzing the wave propagation on the surface of the skin/lung by approximating the boundary of the tissue to be a semi-infinite linear viscoelastic medium under a local harmonic excitation on the surface.

Despite the plethora of models available for lung parenchyma, recent experimental investigations in the application of Rayleigh wave speed to determine the elastic/viscoelastic properties of lung parenchyma have utilized classical/Biot model in their work, and have not considered the effect of pleural membrane tension on Rayleigh waves, except for the work of Man *et al.* in 1991. The work by Man *et al.* [38] provides a technique to measure non-destructively the tension in the pleural membrane utilizing a classical model of the lung parenchyma. It was observed in their work that the predicted values of tension in the pleural membrane were higher than that obtained from the indentation tests. This is possibly due to the porous architecture of the lung. When a lung is inflated, it is observed from histological features and echographic patterns that the porosity and airspace geometry of the lung dramatically changes thereby causing the acoustic behavior of the lung to resemble that of a dry foam [39]. Taking into consideration the observations made above, the present work considers the effect of pleural membrane tension on the propagation of Rayleigh waves in inflated porous lungs by modeling the lung parenchyma as a linear elastic material with voids. The pleural membrane has a considerable effect on the lung's ability to resist both a change of volume and a change of shape [2], [22], [23]. Further, the tension in the membrane limits the propagation of Rayleigh waves to

low-frequencies and hence only low-frequency analysis of the secular equation for Rayleigh waves in a LEMV is considered in the present study. The behavior of low-frequency surface waves in biological tissues has received only limited attention in the literature [40]. The present study, we hope will aid low-frequency surface wave elastographic studies of the lung and will further help non-destructively evaluate the tension in the pleural membrane utilizing the Rayleigh wave speed.

This paper is organized as follows: Section II presents an outline of the theory of LEMV followed by a derivation of the characteristic dispersion equation for low-frequency Rayleigh wave in the parenchyma covered by the pleural membrane. A non-iterative scheme for the determination of a simple root of the dispersion equation is given in Section III. An approximate formula for the Rayleigh wave speed with allowance for porosity and surface effects is derived in Section IV. Section V provides a discussion of the results obtained. The conclusions of the work are given in Section VI.

## II. THEORY

The theory of linear elastic materials with voids [41] is the simplest extension of the classical theory of elasticity, in which, the basic idea is to suppose that there is a distribution of vacuous pores or voids distributed throughout the elastic body. In this theory, the void volume is included as an additional kinematic variable, and in the limiting case of vanishing of this volume, the theory reduces to the classical elasticity theory [41]. The behavior of plane harmonic waves in a LEMV is analyzed in [42], where in it was observed that there are two dilatational waves, one is predominantly the dilatation wave of classical linear elasticity and the other is predominantly a wave carrying a change in the void volume fraction. A study on the propagation of longitudinal and shear waves in an elastic medium with vacuous pores is carried out in [43]. It was shown in [44] that the presence of voids endows the material of LEMV with viscoelastic features akin to that of a standard linear solid. These characteristics of LEMV will be of use in the modeling of biological tissues such as the lung parenchyma. We consider the propagation of the surface waves on an inflated lung at a given transpulmonary pressure (ptp).

The lung parenchyma is assumed to be a homogeneous and isotropic elastic material with voids and the pleura covering the lung as a taut elastic membrane in contact with the parenchyma. We choose a cartesian coordinate system under which the material with voids occupies the region  $x_3 > 0$  and the boundary plane  $x_3 = 0$  is a material surface representing the elastic membrane. The assumption of homogeneity and isotropy for the pleural membrane entails that its residual stress is a constant surface tension [38]. Ignoring gravitational forces, we assume that the membrane is subjected to no external forces other than that acted upon it by the elastic half-space modeled by a material with voids. We further assume that,  $T$  is the constant surface tension and  $\sigma$  is the mass per unit area of membrane in the reference configuration.

In the context of the theory formulated in [41], the field equations for a homogeneous and isotropic material with voids, in the absence of body forces is

$$\mu \nabla^2 U + (\lambda + \mu) \nabla \nabla \cdot U + \beta \nabla \phi = \rho \frac{\partial^2 U}{\partial t^2} \quad (1)$$

$$\alpha \nabla^2 \phi - \xi \phi - \omega \frac{\partial \phi}{\partial t} - \beta \nabla \cdot U = \rho k \frac{\partial^2 \phi}{\partial t^2} \quad (2)$$

where  $U$  is the displacement vector,  $\phi$  is the so-called volume fraction field,  $\lambda, \mu$  are the Lamé's constants,  $\rho$  is the mass density and  $\alpha, \beta, \omega$  and  $k$  are the material constants describing the presence of voids and  $t$  is the time [42]. The methodology of solving the equation for surface wave propagation in a material of LEMV is outlined in [45], [46] and is adopted in the present work. Following it if we set  $U = \text{grad } p + \text{curl } q$  and assume a plane deformation parallel to the  $x_1 x_3$  plane, then on using (1) the functions  $p, q$  satisfy:

$$\left( \nabla^2 - \frac{1}{v_1^2} \frac{\partial^2}{\partial t^2} \right) p = \frac{-\beta}{\lambda + 2\mu} \phi \quad (3)$$

$$\left( \nabla^2 - \frac{1}{v_2^2} \frac{\partial^2}{\partial t^2} \right) q = 0 \quad (4)$$

where  $v_1^2 = \frac{\lambda + 2\mu}{\rho}, v_2^2 = \frac{\mu}{\rho}$

There are three unknown functions  $p, q, \phi$  connected through (2)-(4). The function  $\phi$  can be eliminated from (2), (3) yielding

$$\left\{ \left[ \nabla^2 - \frac{1}{v_1^2} \frac{\partial^2}{\partial t^2} \right] \left[ \nabla^2 - \frac{1}{\alpha^*} \left( 1 + \omega^* \frac{\partial}{\partial t} + k^* \frac{\partial^2}{\partial t^2} \right) \right] + \beta^* \nabla^2 \right\} p = 0 \quad (5)$$

where  $\alpha^* = \frac{\alpha}{\xi}, \omega^* = \frac{\omega}{\xi}, k^* = \frac{\rho k}{\xi}, \beta^* = \frac{\beta^2}{\alpha(\lambda + 2\mu)}$  Microscopic considerations, generally, predict the presence of surface stress whenever a new surface is created. The presence of surface stress results in a non-classical boundary condition which gives the surface traction on the substrate in terms of surface stress and inertia [47].

The boundary conditions [46], [47] are

$$\tau_{i3} + S_{im,m} = \sigma \frac{\partial^2 u_i}{\partial t^2} \text{ for } x_3 = 0 \quad (6)$$

and

$$\frac{\partial \phi}{\partial z} = 0 \text{ on } x_3 = 0 \quad (7)$$

where  $\tau_{ij}$  is the stress tensor in the material of LEMV and  $S_{im}, i = 1, 2, 3; m = 1, 2$  is the surface stress tensor with the normal summation convention being used and a subscript preceded by a comma indicates differentiation with respect to the corresponding coordinate.

$$\text{Here } \tau_{ij} = \lambda \delta_{ij} u_{k,k} + \mu (u_{i,j} + u_{j,i}) + \beta \phi \delta_{ij} \quad (8)$$

$$S_{im} = \delta_{im} T (1 + u_{t,t}) - T u_{m,i} \text{ for } i = 1, 2 \quad (9)$$

$$S_{3m} = T u_{3,m} \quad (10)$$

We seek solutions of (4), (5) in the form of plane waves propagating along the  $x_1$  axis and decaying exponentially into the depth of the medium.

$$\text{Assume } p = f(x_3) \exp(i(k_1 x_1 - \omega_1 t)) \quad (11)$$

$$q = g(x_3) \exp(i(k_1 x_1 - \omega_1 t)) \quad (12)$$

where  $\omega_1$  is a known angular frequency,  $f(x_3), g(x_3)$  are functions of  $x_3$  tending to zero as  $x_3 \rightarrow \infty$  and  $k_1$  is the wave number. Equations (11), (12) correspond to Rayleigh waves propagating with wavelength  $2\pi/k_1$  and phase velocity  $c = \omega_1/k_1$ .

Using (11), (12) in (4), (5) and the boundary conditions (6), (7) we obtain a characteristic equation for Rayleigh waves in a LEMV under surface effects. As we are interested in a better understanding of the surface wave behavior on biological tissue in the low audible frequency regime, we retain the lowest degree terms in  $\omega_1$  in the characteristic equation. The characteristic equation for low frequency Rayleigh waves in the lung parenchyma modeled by a LEMV and covered by a taut membrane having a certain mass and a constant surface tension is

$$\begin{aligned} (2 - s^2)^2 - 4(1 - s^2)^{\frac{1}{2}} (1 - s^2 \tau_0^2)^{\frac{1}{2}} \\ = \frac{k_1 s^2}{\mu} (1 - s^2 \tau_0^2)^{1/2} (T - \sigma c^2) \end{aligned} \quad (13)$$

where

$$\tau_0^2 = \frac{v_2^2}{v_1^2(1 - N)} = \theta \text{ (say); } s^2 = \frac{c^2}{v_2^2} \quad (14)$$

Here  $N$  is the dimensionless coupling number defined by [41]

$$N = \frac{\beta^2}{\xi(\lambda + 2\mu)}, \quad 0 \leq N < 1, \xi > 0, \mu > 0 \quad (15)$$

where  $\beta, \xi$  are parameters corresponding to the void volume fraction field,  $\xi$  is a material modulus that handles the void stiffness and provides information relating to the void strength while  $\beta$  is a material constant that bridges the micro-dilatation effects to the macro-deformation effects [48]. If  $\sigma = 0$ , (13) reduces to the characteristic equation for low frequency Rayleigh waves in a LEMV under the action of surface stress [46]. If  $\sigma = 0, N = 0$ , (13) reduces to the dispersion equation for Rayleigh waves with allowance for surface tension [49]. If  $T = 0, \sigma = 0, N = 0$ , (13) reduces to the classical Rayleigh wave equation [8]. Using (13), we study the influence of surface tension in the pleural membrane and the porous structure of the lung parenchyma on the propagation of Rayleigh waves in inflated lungs.

### III. NUMERICAL SCHEME

A simple method of finding a numerical approximation to a root of the characteristic equation (13) is not only desirable but essential. A simple non-iterative method due to Ioakimidis and Anastasselou [50] is utilized here for the determination of a simple root of the secular equation (13) for low frequency Rayleigh waves in the parenchyma modeled

by a LEMV. The method is based on the computation of an integral involving the function whose simple root along a finite interval is to be determined. The integral is evaluated utilizing Gauss- and Lobatto- Chebyshev quadrature rules for regular intervals and equating the obtained results.

Let us assume that  $f(x)$  is an algebraic or transcendental function that possesses a simple root  $c$  in a finite interval  $(a, b)$  and it is assumed that  $f(x)$  is continuous in  $[a, b]$  and has continuous second derivative in a neighborhood of  $c$ .

Consider the integral  $I = \int_a^b \frac{1}{\sqrt{(b-x)(x-a)}} \frac{x-c}{f(x)} dx$  (16)

The value of  $c$  is determined approximately by evaluating the integral  $I$  utilizing Gauss- and Lobatto- Chebyshev quadrature rules for regular intervals in  $[-1, 1]$ .

The quadrature rule takes the forms given by [50]

$$\int_a^b \frac{g(x)}{\sqrt{(b-x)(x-a)}} dx = \frac{\pi}{n} \sum_{i=1}^n g(x_i) + E_n \quad (17)$$

$$\int_a^b \frac{g(x)}{\sqrt{(b-x)(x-a)}} dx = \frac{\pi}{n} \sum_{i=1}^{n'} g(x_i^*) + E_n^* \quad (18)$$

where  $x_i = \frac{(a+b)-(a-b)t_i}{2}, i = 1, 2, \dots, n$  with

$$t_i = \cos \frac{(2i-1)\pi}{2n}, \quad i = 1, 2, \dots, n$$

$x_i^* = \frac{(a+b)-(a-b)t_i^*}{2}, i = 0, 1, \dots, n$  with  $t_i^* = \cos \frac{i\pi}{n}, i = 0, 1, \dots, n$

Utilizing (17) and (18) for the evaluation of integral (16) and equating the results and further assuming that the error terms rapidly approach zero as  $n \rightarrow \infty$ , we have the following approximation  $c_n$  to  $c$  given by

$$c \approx c_n = \frac{\sum_{j=0}^{2n'} \frac{(-1)^j y_j}{f(y_j)}}{\sum_{j=0}^{2n''} \frac{(-1)^j}{f(y_j)}} \quad (19)$$

where  $y_j = \frac{(a+b)-(a-b)u_j}{2}$  and  $u_j = \cos \left(\frac{j\pi}{2n}\right), j = 0, 1, \dots, 2n$ .

Here double prime in summation means that the first and the last term of the sum is halved. The formula has earlier been used to obtain a root of the secular equation of Rayleigh waves in isotropic and anisotropic elastic solids [51].

#### IV. PLEURAL MEMBRANE EFFECTS AND RAYLEIGH WAVES: APPROXIMATE FORMULA

In this section, we consider the Rayleigh waves propagating at the pleural membrane represented by the plane material boundary of an elastic half space containing a distribution of voids that models the lung parenchyma. We consider a smooth contact between the two, ignore gravitation and assume that the pleural membrane is subject to no external forces other than that acted upon it by the half-space. The waves are generally dispersive and the dispersion is caused by both the presence of pleural membrane and the porous architecture of lung parenchyma.

We first solve (13) using perturbation method and derive an approximate formula for the Rayleigh wave velocity in the parenchyma with allowance for surface tension, mass per unit area of membrane and the void coupling parameter. Equation (13) can be written as

$$F(\gamma) = \frac{k_1 \gamma}{\mu} (1 - \gamma\theta)^{1/2} (T - \sigma c^2) \quad (20)$$

where  $F(\gamma) = (2 - \gamma)^2 - 4(1 - \gamma)^{1/2} (1 - \gamma\theta)^{1/2}$  with  $\gamma = s^2$

Utilizing the expression for  $k_1, c^2, s^2$  (20) can be expressed as

$$F(\gamma) = \frac{\omega_1 T}{\mu v_2} \gamma^{1/2} (1 - \gamma\theta)^{1/2} - \frac{\omega_1 \sigma v_2}{\mu} \gamma^{3/2} (1 - \gamma\theta)^{1/2}$$

Denoting  $b_1(\omega_1) = \frac{\omega_1 T}{\mu v_2}$  and  $b_2(\omega_1) = \frac{\omega_1 \sigma v_2}{\mu}$

$$\text{We have } F(\gamma) = b_1(\omega_1) \gamma^{1/2} (1 - \gamma\theta)^{1/2} - b_2(\omega_1) \gamma^{3/2} (1 - \gamma\theta)^{1/2} \quad (21)$$

With  $T = \sigma = 0$  (21) reduces to

$$F(\gamma) = 0 \quad (22)$$

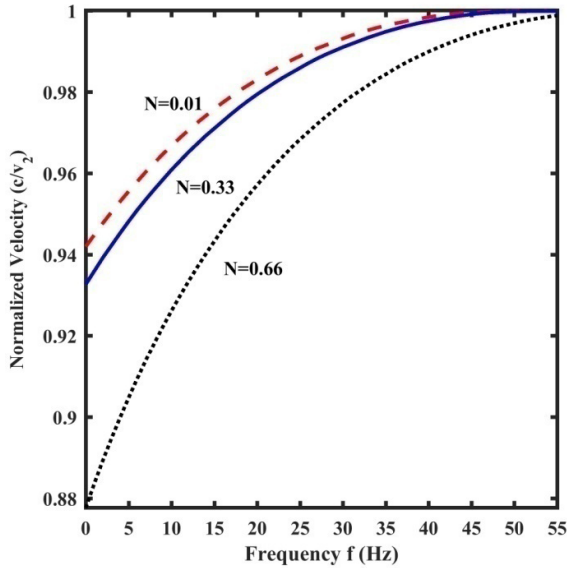
which is the equation describing the velocity of low frequency Rayleigh waves in a LEMV. For a fixed  $N$ , let  $\gamma_0$  be a root of (22). For the same  $N$ , we solve (21) by the perturbation method, writing the solution in the form  $\gamma = \gamma_0 + \gamma_1$ , where  $\gamma_1$  is a small addition caused by the surface tension and mass per unit area of the membrane. Substituting  $\gamma = \gamma_0 + \gamma_1$  in (21) and neglecting the terms of the second order in  $\omega$  and  $\gamma_1$  we obtain after a lengthy calculation

$$\begin{aligned} \gamma_1 &= \frac{\gamma_0^{1/2} (1 - \gamma_0\theta)^{1/2}}{F'(\gamma_0)} (b_1(\omega_1) - \gamma_0 b_2(\omega_1)) \\ &= \frac{\gamma_0^{1/2} (1 - \gamma_0\theta)^{1/2}}{F'(\gamma_0)} \left( \frac{\omega_1 T}{\mu v_2} - \gamma_0 \frac{\omega_1 \sigma}{\mu} v_2 \right) \\ \gamma_1 &= \frac{\omega_1 \gamma_0^{1/2} (1 - \gamma_0\theta)^{1/2}}{F'(\gamma_0) v_2} \left( \frac{T}{\mu} - \gamma_0 \frac{\sigma}{\rho} \right) \end{aligned} \quad (23)$$

In (23)  $T, \sigma$  are the parameters of the membrane, while  $\theta, v_2$  are the parameters related to the parenchyma. For fixed values of these parameters, it is seen that  $\gamma_1$  depends on the frequency  $f$ . The approximate final expression for the Rayleigh wave velocity ( $c$ ) with regard for the surface tension ( $T$ ) and mass per unit area ( $\sigma$ ) of the pleural membrane is given by

$$\begin{aligned} \frac{c}{v_2} &= \gamma^{1/2} = (\gamma_0 + \gamma_1)^{1/2} \approx \gamma_0^{1/2} \left( 1 + \frac{1}{2} \frac{\gamma_1}{\gamma_0} \right) \\ \frac{c}{v_2} &\approx \gamma_0^{1/2} \left( 1 + \frac{\gamma_1}{2\gamma_0} \right) \end{aligned} \quad (24)$$

where  $\gamma_1$  is given by (23). By performing experiments such as those outlined in [9]–[13] at a fixed frequency, one can use (24) to determine with sufficient accuracy the variations of surface tension in the pleural membrane under the influence of various other factors. In addition, measurements at several frequencies would in principle, provide information for non-destructively estimating the value of surface tension.

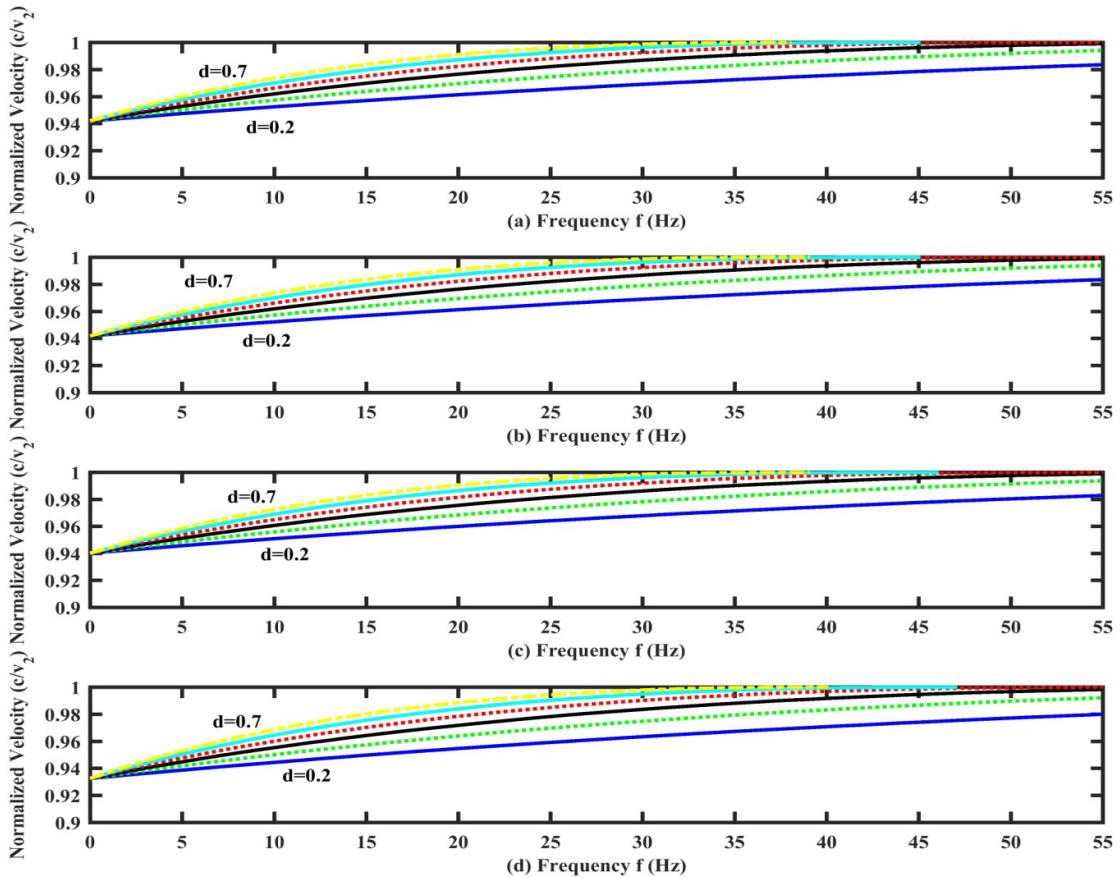


**FIGURE 1.** Dispersion curves of Rayleigh waves in a typical dog lung for different values of coupling parameter (N).

**V. NUMERICAL RESULTS AND DISCUSSION**

For a numerical experiment, we consider the typical value of the dog lung at ptp of 5 cm  $H_2O$  [45]. The values are

$\frac{T}{\mu} = \frac{1}{2}cm, \frac{\mu}{\lambda+2\mu} = \frac{1}{6}; \mu = 4000dynes/cm^2; \sigma = 3 \times 10^{-3}gm/cm^2$  and  $\rho = 0.2 gm/cc$ . The values are used in (24) to obtain the approximate value of  $\frac{c}{v_2}$  where  $c$  is the velocity of propagation of Rayleigh waves. The values obtained for different values of the coupling parameter  $N$  and for different frequencies are given in Table 1. The presence of a sufficiently high tension in the pleural membrane will lead to the existence of a cutoff frequency ( $f_0$ ). No Rayleigh-type wave that has frequency ( $f > f_0$ ) and has a real phase velocity ( $c$ ) may propagate along the surface of the inflated lung [38]. The cut-off frequency is influenced by both the porous structure of the parenchyma and the surface tension in the membrane. These two parameters determine the pathology of the lung and we first consider the influence of the pores/voids in the parenchyma through the coupling number ( $N$ ) on the cut-off frequency values and then study the effect of pleural membrane on the propagation of low frequency Rayleigh waves in inflated lungs through the dispersion curves. In the low frequency regime, as seen from (13), the presence of voids is reflected in the coupling number  $N$  and this number plays a crucial role in the existence of Rayleigh waves for the whole range of Poisson’s ratio  $\nu \in [-1, 0.5]$ . From (13), it is seen that for the existence of Rayleigh waves with the real



**FIGURE 2.** Dispersion curves of Rayleigh waves for varying values of  $d = \frac{T}{\mu} = 0.2, 0.3, 0.4, 0.5, 0.6, 0.7$  in the pleural membrane with (a)  $N = 0$  (b)  $N = 0.01$  (c)  $N = 0.1$  (d)  $N = 0.33$ .

**TABLE 1. Approximate values of Normalized Rayleigh wave velocity in Porous dog lungs.**

Coupling Parameter	$f(\text{Hz})$	$(\gamma_0)^{1/2}$	$\gamma_1$	$\frac{c}{v_2} = \gamma_0^{1/2} \left(1 + \frac{\gamma_1}{2\gamma_0}\right)$	Exact value
$N=0$	1	0.942241882233894	0.005517494011758	0.944549687119458	0.945093537801564
	4	0.942241882233894	0.022069976047030	0.951473101776150	0.952987751884692
	8	0.942241882233894	0.044139952094061	0.960704321318406	0.962344820196278
	12	0.942241882233894	0.066209928141091	0.969935540860662	0.970142968533097
	16	0.942241882233894	0.088279904188121	0.979166760402919	0.976900866840715
$N=0.01$	1	0.942072802867109	0.005530722404043	0.944384895680428	0.944933205747231
	4	0.942072802867109	0.022122889616171	0.951321174120382	0.952845492894599
	8	0.942072802867109	0.044245779232343	0.960569545373656	0.962225383105381
	12	0.942072802867109	0.066368668848514	0.969817916626929	0.970044217894383
	16	0.942072802867109	0.088491558464686	0.979066287880202	0.976816043118280
$N=0.33$	1	0.932661990676125	0.006267065768286	0.935204175319008	0.935989256267380
	4	0.932661990676125	0.025068263073143	0.942830729247657	0.945109959165068
	8	0.932661990676125	0.050136526146286	0.952999467819189	0.955595825418794
	12	0.932661990676125	0.075204789219430	0.963168206390722	0.964637458214488
	16	0.932661990676125	0.100273052292573	0.973336944962254	0.972159877649631

**TABLE 2. Cutoff frequency values for different values of coupling parameter (N).**

$N$	0	0.01	0.1	0.2	0.3	0.4	0.5	0.6
$f_0(\text{Hz})$	50.817	50.868	51.391	52.137	53.145	54.586	55.445	60.737

phase velocity, we have

$$\frac{1 - 2\nu}{2(1 - \nu)(1 - N)} < 1 \tag{25}$$

For the soft biological tissues where  $\nu \in [0.4, 0.5]$ , we have  $N \leq 0.66$ . If we let  $\omega_1 = 2\pi f$  where  $f$  is the frequency then from (13), we get

$$f = \frac{\left(2 - \frac{c^2}{v_2^2}\right)^2 - 4\sqrt{1 - \frac{c^2}{v_2^2}}\sqrt{1 - \frac{c^2}{v_1^2(1-N)}}}{\sqrt{1 - \frac{c^2}{v_1^2(1-N)}}(T - \sigma c^2)2\pi\rho\left(\frac{c}{v_2^4}\right)} \tag{26}$$

As  $c \rightarrow v_2$ , the value approached by  $f$  is denoted  $f_0$ , the cut-off frequency. As seen from (26), the cut off frequency at which surface excitation establishes the propagation of Rayleigh waves depends on the tension in the membrane and the mechanical properties of the parenchyma. The cut-off frequency values for different values of  $N$  are given in Table 2. We observe that as  $N$  increases the cut-off frequency values increases. From (15) we see that increasing  $N$  means that the ratio  $\frac{\beta^2}{\xi}$  increases i.e. when the ratio of the micro-dilatation effects to the macro-deformation effects increase, the cut-off frequency value increases. In terms of diagnostics, when the cut-off frequency value is approached then for known density of parenchyma, the shear modulus of the parenchyma is obtained. Further the measurement of  $f_0$  gives an estimate of surface tension  $T$  in the pleural membrane. We now utilize the numerical scheme outlined in Section III to solve (13) to obtain the dispersion spectrum. The dispersion curves

pertaining to  $N = 0.01, 0.33$  and  $0.66$  are depicted in Fig 1. The frequency  $f$  (in Hz) is taken along the  $x$ - axis and the normalized velocity  $(c/v_2)$  is taken along the  $y$ - axis. From the dispersion curves, we observe that the porous structure of the parenchyma decreases the Rayleigh wave velocity, which is as expected. An increasing  $N$  is reflected by an increase in  $\beta^2/\xi$  that is instrumental in increasing the spongy nature of the medium i.e. the flexibility of the voids against the macro-response of the lung. The spongy nature of the lung is found to increase the cut-off frequency values and decrease the Rayleigh wave velocity. Further, since  $T$  increases with species size whereas  $\mu$  does not [22], we observe the influence of tension on the dispersion of Rayleigh waves by varying the values of  $\frac{T}{\mu}$  for different values of  $N$ . The dispersion curves for  $d = \frac{T}{\mu} = 0.2, 0.3, 0.4, 0.5, 0.6$  and  $0.7$  and  $N = 0.0, 0.01, 0.1, 0.33$  are given in Fig 2. For different values of  $d = \frac{T}{\mu}$  ranging from 0.2 to 0.7 the dispersion curves are illustrated for each  $N$ . The frequency values are taken along the  $x$ - axis and the normalized velocity value are taken along  $y$ - axis. With increasing  $f$  the decrease in Rayleigh wave velocity is noticeable for increasing  $N$ . For a fixed  $N$ , with increasing value of  $\frac{T}{\mu}$ , the velocity is found to increase.

The effect of the pleura membrane and voids in the parenchyma on the propagation of Rayleigh waves on inflated lungs is brought out in the figures. In terms of diagnostics, the dispersion curves can be used to non-destructively estimate the tension in the pleural membrane in relation to the porous architecture of lung parenchyma once the surface wave speed is obtained.

## VI. CONCLUSION

The frequency dependence of the pleural contribution to the lung behavior is examined in this paper. The effect of natural porosity of the lung parenchyma is included in the study by modeling the parenchyma as a linear elastic material with voids. An approximate formula for Rayleigh wave velocity with allowance for pleural membrane effects and porous architecture of the lung has been derived using the perturbation technique. The formula is likely to aid experimenters determine the variations in surface tension of the membrane under the influence of other factors at a fixed frequency and at a fixed value of the coupling parameter. In addition, the cutoff frequency at which surface excitation establishes propagation of Rayleigh waves are obtained for different values of the coupling parameter. The role of the pleural membrane and the porous architecture of lung parenchyma are illustrated through the dispersion curves and are likely to aid studies on compressive atelectasis in the lung.

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